**Lecture 16 General pathology Dr. Ali H. Murad**

**Systemic pathology**

***PATHOLOGY OF THE GIT***

## THE ORAL CAVITY & OROPHARYNX

Many pathological processes can affect the constituents of the oral cavity.

**1-PROLIFERATIVE LESION**; Irritation fibroma , ossifying fibroma, pyogenic granuloma , peripheral giant cell granuloma

**2**-**INFLAMMATERY ULCERATION**; traumatic ,aphthous

**3**-**INFECTIONS ;**Herpes simplex infections, oral candidiasis (thrush) ,deep Fungal Infections .

## THE TUMOR AND PREMALIGNANT LESIONS

***Leukoplakia***  is a white patch that cannot be scraped off and cannot be attributedclinically or microscopically to any other disease i.e. if a white lesion in the oral cavity can be given a specific diagnosis it is not a leukoplakia. As such, white patches caused by entities such as candidiasis are not leukoplakias. *All leukoplakias must be considered precancerous (have the potential to progress to squamous cell carcinoma) until proved otherwise through histologic evaluation.*

***Erythroplakias*** are red velvety patches that are much less common, yet much more serious than leukoplakias. The incidence of dysplasia and thus the risk of complicating squamous cell carcinoma is much more frequent in erythroplakia compared to leukoplakias. Both leukoplakia and erythroplakia are usually found between ages of 40 and 70 years, and are much more common in males than females. The use of tobacco (cigarettes, pipes, cigars, and chewing tobacco) is the most common incriminated factor.

### Squamous cell carcinoma

The vast majority (95%) of cancers of the head and neck are squamous cell carcinomas; these arise most commonly in the oral cavity.

## ESOPHAGUS

The main functions of the esophagus are to:

1. Conduct food and fluids from the pharynx to the stomach
2. Prevent reflux of gastric contents into the esophagus.

These functions require motor activity coordinated with swallowing.

## CONGENITAL ANOMALIES

Several congenital anomalies affect the esophagus including:

1-the presence of ***ectopic*** gastric mucosa & pancreatic tissues within the esophageal wall.

2-***congenital cysts*** *&****congenital herniation*** of the esophageal wall into the thorax.

3-The ***atresia****,* a segment of the esophagus is represented by only a noncanalized cord, with the upper pouch connected to the bronchus or the trachea and a lower pouch leading to the stomach.

4***-Mucosal webs*** are shelf-like, eccentric protrusions of the mucosa into the esophageal lumen. These are most common in the upper esophagus. The triad of upper esophageal web, iron-deficiency anemia, and glossitis is referred to as ***Plum mer-Vinson syndrome***.

1. ***Esophageal rings*** unlike webs are concentric plates of tissue protruding into the lumen of the distal esophagus. Episodic dysphagia is the main symptom

1. ***Stenosis*** consists of fibrous thickening of the esophageal wall. Although it may be congenital, it is more frequently the result of severe esophageal injury with inflammatory scarring, as from gastroesophageal reflux disease (GERD), radiation, scleroderma and caustic injury. Stenosis usually manifests as progressive dysphagia , at first to solid food but eventually to fluid as well.

# Esophagitis

This term refers to inflammation of the esophageal mucosa. It may be caused by a variety of physical, chemical, or biologic agents.

***Reflux Esophagitis (Gastroesophageal Reflux Disease or GERD):*** is the most important cause of esophagitis and signifies esophagitis associated with reflux of gastric contents into the lower esophagus. The action of gastric juices is vital to the development of esophageal mucosal injury.

## *-Gross (endoscopic) features*

These depend on the causative agent and on the duration and severity of the exposure.

Mild esophagitis may appear grossly as simple hyperemia. In contrast, the mucosa in severe esophagitis shows confluent erosions or total ulceration into the submucosa.

## *-Microscopic features*

Three histologic features are characteristic:

1. Inflammatory cells including eosinophils within the squamous mucosa.
2. Basal cells hyperplasia
3. Extension of lamina propria papillae into the upper third of the mucosa. The clinical manifestations consist of dysphagia, heartburn, regurgitation of a sour fluid into the mouth, hematemesis, or melena. Rarely, there are episodes of severe chest pain that may be mistaken for a "heart attack."

**TUMORS**

**Benign Tumors**

**Leiomyomas**are the most common benign tumors of the esophagus.

## Malignant Tumors

Carcinomas of the esophagus (5% of all cancers of the GIT) have, generally, a poor prognosis because they are often discovered too late. Worldwide, squamous cell carcinomas constitute 90% of esophageal cancers, followed by adenocarcinoma.

Other tumors are rare.

**STOMACH**

In developed countries, peptic ulcers occur in up to 10% of the general population. Chronic infection of the gastric mucosa by the bacterium Helicobacter pylori is the most common infection worldwide. Gastric cancer is still a significant cause of death, despite its decreasing incidence.

**GASTRITIS** this is by definition, "*inflammation of the gastric mucosa*". It is a microscopic diagnosis**.** The inflammation may be acute, with neutrophilic infiltration, or chronic, with lymphocytes and/or plasma cells.

**Acute gastritis** is usually transient in nature. The inflammation may be accompanied by hemorrhage into the mucosa (*acute hemorrhagic gastritis***)** and, sometimes by sloughing (erosions) of the superficial mucosa (*acute erosive gastritis*). The latter is a severe form of the disease & an important cause of acute gastrointestinal bleeding.

Although a large number of cases have no obvious cause (idiopathic), acute gastritis is frequently associated with

1. *Heavy use of nonsteroidal anti-inflammatory drugs* (NSAIDs)*,* particularly aspirin, cancer chemotherapeutic drugs, or radiation
2. *Excessive consumption of alcohol, heavy smoking, and ingestion of strong acids or alkali* as in suicidal attempts
3. *Uremia*
4. *Severe stress* (e.g., trauma, burns, surgery)
5. *Mechanical trauma* (e.g., nasogastric intubation)
6. *Distal gastrectomy*(reflux of duodenal contents).

**Chronic Gastritis** is defined as "chronic inflammation of the gastric mucosa that eventuates in mucosal atrophy and intestinal metaplasia". The epithelial changes may progress to dysplasia, which constitute a soil for the development of carcinoma.

The major etiologic associations of chronic gastritis are:

1. Chronic infection by H. pylori
2. autoimmune damage
3. Excessive alcohol consumption & heavy cigarette smoking
4. Post-antrectomy (due to reflux of bile-containing duodenal secretions)
5. Outlet obstruction, uremia, and other rare causes

## Autoimmune gastritis

About 10% of chronic gastritis are autoimmune in nature. It results from the presence of autoantibodies to components of parietal cells, including the acidproducing enzyme H+/K+-ATPase, gastrin receptor, and intrinsic factor. Gland destruction and mucosal atrophy lead to loss of acid production (hypo- or achlorhydria). In the most severe cases, production of intrinsic factor is also impaired, leading to pernicious anemia. Affected patients have a significant risk for developing gastric carcinoma and endocrine tumors (carcinoid tumor)

### -Gross (endoscopic) features

The mucosa of the affected regions is usually hyperemic and has coarser rugae than normal.

With long-standing disease, the mucosa may become thinned and flattened because of atrophy.

### -Microscopic features

Irrespective of cause or location, the microscopic changes are similar:

-The mucosa is infiltrated by lymphocytes & plasma cells.

-Frequently the lymphocytes are disposed into aggregates i.e. follicles, some with germinal centers.

-Neutrophils may or may not be present.

### Several additional histologic features are characteristic; these include

- *Intestinal metaplasia*: the mucosa may become partially replaced by metaplastic columnar cells and goblet cells of intestinal morphology; these may display flat or villous arrangement. If the columnar cells are absorptive (with ciliated border) the metaplasia is termed complete, otherwise it is incomplete.

- *Atrophy* as evidecnced by marked loss of the mucosal glands. Parietal cells, in particular, may be absent in the autoimmune form.

-*Dysplasia:* with long-standing chronic gastritis, the epithelium develops dysplastic changes. Dysplastic alterations may become so severe as to constitute in situ carcinoma. *The development of dysplasia is thought to be a precursor lesion of gastric cancer.* It occurs in both autoimmune and H. pylori- associated chronic gastritis.

-In those individuals infected by H. pylori, the organism lies in the superficial mucus layer on the surface and within the gastric pits. They do not invade the mucosa. These bacteria are most easily demonstrated with silver or Giemsa (special) stains.